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Sutural Bone Frequency in Synostotic Rabbit Crania

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ABSTRACT This study tests the hypothesis that crania with synostosed sutures will have a significantly higher incidence of calvarial sutural bones than normal crania. Sutural bones were counted in seven calvarial sutures and compared among four groups of adult New Zealand white rabbit skulls: normal in-colony (NI) controls (N = 14), normal out-colony (NO) controls (N = 12), skulls with familial delayed onset (DO) coronal synostosis (N = 25), and skulls with experimentally immobilized coronal sutures (EI) (N = 20). Comparisons among groups were made with a Kruskal-Wallis one-way ANOVA and between groups with a Mann-Whitney U-test, using a Bonferroni correction for multiple comparisons. Significant differences ($P < 0.05$) were noted only in the coronal and sagittal sutures, with EI crania having the greatest number of coronal sutural bones; between group differences were undetectable for sagittal sutural bones. A post hoc two-sample binomial test for equal proportions showed that the distribution of coronal sutural bones among individuals across groups was even, while the distribution of sagittal sutural bones was significantly higher in EI crania. These results suggest that altered sutural forces of the calvaria contribute to an increased occurrence of sutural bones. However, the influence of inheritance on increased occurrence of sutural bones cannot be discounted, as reflected in the equivalent number of individuals across groups that possessed coronal sutural bones. Am J Phys Anthropol 102:555–563, 1997.

It has been well established that calvarial suture morphology is influenced by and responsive to extrinsic loads on the cranium (Moss, 1954, 1957; Young, 1959; Moss and Young, 1960; Herring, 1972; Jaslow, 1989, 1990; Richards and Antón, 1991). For example, increased calvarial suture complexity (i.e., sutural interdigitation plus sutural bone frequency) has been associated with altered mechanical loading found in hydrocephaly (Pfeiffer, 1900; Young, 1959; Broth-

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well, 1981; Sperber, 1989; Richards and Antón, 1991) and in artificial cranial deformation (Dorsey, 1897; Ossenberg, 1970; Pucciarelli, 1974; Antón et al., 1992; Konigsberg et al., 1993; White, 1996). In both hydrocephaly and artificial cranial deformation, the normal neurocapsular growth vectors are altered, presumably changing the loads on the calvarial sutures and resulting in increased sutural complexity (Young, 1959; Pucciarelli, 1974; Richards and Antón, 1991). This study investigates the role of altered mechanical loading on one component of sutural complexity, sutural bone occurrence (Antón et al., 1992).

Sutural bones (wormian bones, ossicles, or supernumerary bones) are multiple, small, irregularly shaped bones that develop as extra islands of bone within the calvarial sutures of the skull (Brothwell, 1981; Schwartz, 1995). These structures rarely, if ever, appear in the basicranial synchondroses or facial sutures (Hess, 1946; Brothwell, 1981; Schwartz, 1995). Sutural bones represent independent centers of ossification and usually penetrate both the outer and inner tables of the cranial vault (Sperber, 1989; Antón et al., 1992). Sutural bones are not to be confused with Inca bones, which are large triangular bones found at the junction of the occipital and parietal bones. Inca bones are considered to be under strong genetic control and may represent nonfusion of two primary ossification centers of the occipital bone (Antón et al., 1992; Schwartz, 1995).

Sutural bones are a normal occurrence in many mammalian taxa (Green and Fekete, 1932; Grüneberg, 1961; Berry and Searle, 1963), including nonhuman primates (Delattre et al., 1969; Cheverud and Buikstra, 1981; Richtsmeier et al., 1984) and humans (Sullivan, 1922; Bennett, 1965; Berry and Berry, 1967; Kellock and Parsons, 1970; Ossenberg, 1970; Trinkaus, 1978). Although sutural bones commonly occur in both normal and pathological crania, their etiology and function (if any) remain unclear. Hess (1946) asserted that sutural bones are formed as a result of a change in the normal ossification of the mesodermal skull membrane due to a metabolic disorder. Little supporting evidence for this cause has been found.

An underlying genetic predisposition for sutural bone development has been proposed, with epigenetic biomechanical factors having only secondary effects, if any, on their appearance (Torgerson, 1951, 1954; Berry and Berry, 1967; El-Najjar and Dawson, 1977). However, other investigators have proposed a primary role to epigenetic stresses and loads on the calvarial sutures, such as those created by artificial deformation (Dorsey, 1897; Dembo and Imbelloni, 1938; Bennett, 1965). This proposed etiology is also supported by experimental studies which have created sutural bones in animal models by altering mechanical load vectors on the cranium (Moss, 1954; Young, 1959; Pucciarelli, 1974). Other authors suggest that sutural bone appearance is a genetic event with epigenetic loading responsible for increases in their frequencies (Ossenberg, 1970; Antón et al., 1992; Konigsberg et al., 1993; White, 1996). Sutural bones have been observed in colonies of rabbits with familial coronal suture synostosis (Greene, 1933; Greene and Brown, 1932; Mooney et al., 1994a; Burrows, 1995) and have been proposed to be a stage in the synostotic progression.

While many studies have indeed revealed an increase in sutural bone frequency associated with artificial cranial deformation (Dorsey, 1897; Dembo and Imbelloni, 1938; Ossenberg, 1970; Antón et al., 1992; Konigsberg et al., 1993; White, 1996), some studies have found no apparent relationship between the two (Sullivan, 1922; El-Najjar and Dawson, 1977). The lack of consensus regarding sutural bone frequency and artificial cranial deformation may be due to several factors. Many studies on sutural bones and deformation have been qualitative (Dorsey, 1897; Sullivan, 1922; Dembo and Imbelloni, 1938). Some studies lumped different deformational methods into a single "deformed" category (Dorsey, 1897; El-Najjar and Dawson, 1977), thus not taking into account the different intensities and stress vectors produced by differing immobilization techniques.

As suggested by Pucciarelli (1974), when the comparative method gives variable results, one must resort to experimental models. This study examines the relationship between altered mechanical loading of the calvaria and sutural bone frequency in a colony of rabbits with familial delayed onset

coronal suture synostosis (Burrows et al., 1994; Mooney et al., 1993, 1994a,b, 1996a) and in rabbits with experimentally produced coronal suture immobilization (Losken et al., 1991). Craniosynostosis, or premature calvarial suture fusion, may occur in any one or a combination of calvarial sutures (Cohen, 1986, 1993). Its etiology is unclear and heterogenous but may occur either as a genetic event (Cohen, 1977), an epigenetic event (Graham et al., 1980; Koskinen-Moffett, 1986; Koskinen-Moffett and Moffett, 1989), or in association with other primary malformations such as hyperthyroidism (Menking et al., 1972), rickets (Coleman and Foote, 1954; McCarthy and Reid, 1980), or hydrocephaly (Andersson, 1966; Kloss, 1986). Delayed onset coronal synostosis is a familial condition seen in both humans and in this colony of rabbits wherein the coronal suture grows significantly less than normal, with osseous bridging occurring across the suture. This bridging prevents normal growth rates at the coronal suture and also alters growth at other calvarial sutures (Reddy et al., 1990; Cohen et al., 1993; Mooney et al., 1994b, 1996b). The experimentally immobilized group represents an artificially created coronal synostosis where genetic events would have no role in the resultant calvarial morphology (Lalikos et al., 1995).

This study tests genetic and epigenetic hypotheses of increased sutural bone frequency in homogenetic rabbits with familial delayed onset and experimentally created coronal suture synostosis. If increased frequency of sutural bones is epigenetically regulated, then both crania with experimental coronal suture immobilization and delayed onset coronal synostosis will have significantly more sutural bones than normal crania. If the increased frequency is genetically regulated, however, crania with delayed onset synostosis will have significantly more sutural bones than both experimentally immobilized and normal crania.

MATERIALS AND METHODS

Skulls from 71 adult New Zealand white rabbits (*Oryctolagus cuniculus*) were used in this study. Twenty-five of the rabbits had congenital delayed onset coronal craniosynostosis (DO), 14 were normal in-colony controls (NI), 12 were normal out-colony controls (NO), and 20 were out-colony rabbits with experimental coronal suture immobilization (EI). Males and females were randomly selected, as New Zealand white rabbits show little sexual dimorphism in body size (Fox, 1994) or synostosis of the coronal suture (Mooney et al., 1994a,b).

All in-colony rabbits were bred in the animal vivarium of the Anthropology Department at the University of Pittsburgh. All out-colony rabbits were obtained from one local breeding facility. Animals were housed in the Anthropology animal vivarium and supplied with an ad libitum amount of water. A veterinarian-prescribed diet of Agway ProLab high fiber rabbit food was provided, as well as regular supplements of timothy and alfalfa hay, apples, and carrots. Temperatures in the vivarium were maintained between 20 and 22°C. All experimental manipulations were approved by the Institutional Animal Care and Use Committee, University of Pittsburgh.

In order to determine which in-colony rabbits grew normally at the coronal suture and which had delayed onset synostosis, all rabbits had radiopaque silver amalgam markers implanted on both sides of the coronal suture at bregma at 10 days of age (Fig. 1). Dorsal and lateral radiographs were taken at 10 days and 6 weeks of age by anesthetizing each rabbit with an intramuscular injection of a solution of 91% Ketaset (ketamine hydrochloride) and 9% Rompun (xylazine) (0.59 mL/kg). Each rabbit was placed in the cephalostat, and their heads were immobilized in a prone position in a styrofoam head holder which was specially designed for each age, with the rostrum and palate parallel to the floor (Mooney et al., 1994b). All radiographs were taken with a Philips Oralix 70A dental x-ray unit with exposures of 50 kV and 7 mA and exposure times between 0.17 and 0.45 seconds. A constant distance of 152 cm from the cassette tube to the cranium was maintained in all radiographs. Growth differences between both right and left sets of suture markers were assessed by visually locating and identifying the markers and computing the increase in distance between the initial 10 day

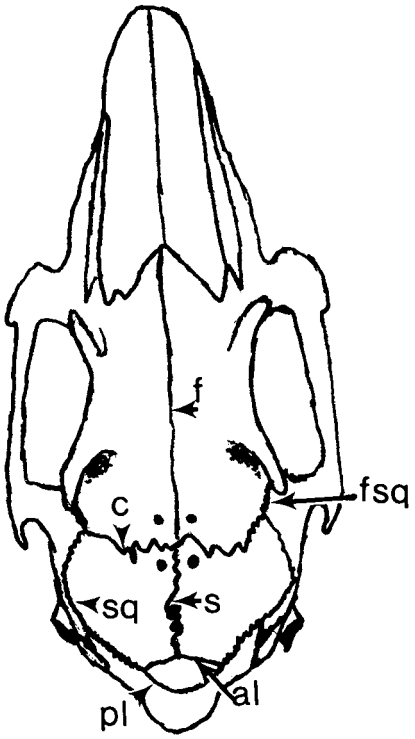

Fig. 1. Dorsal view of rabbit skull showing placement of the amalgam markers (solid black dots) and the seven calvarial sutures assessed for sutural bone presence. al, anterior lambdoid suture; c, coronal suture; f, frontal suture; fsq, frontosquamosal suture; pl, posterior lambdoid suture; s, sagittal suture; sq, squamosal suture.

and the 6 week radiograph. All rabbits who were below a 95% confidence interval for normal growth at the coronal suture, based upon data from out-colony rabbits (Losken et al., 1991), were included in the delayed onset group (Mooney et al., 1994b); all other rabbits were placed in the normal group.

Genetic differences alone could produce significant differences in sutural bone occurrence between delayed onset craniosynostotic rabbits and normal rabbits. To control for this, normal out-colony rabbits with experimentally immobilized coronal sutures were also used. Methylmethacrylate adhesive was placed bilaterally across the coronal sutures at 10 days of age, at the same time that amalgam markers for radiographic data collection were implanted, using the methods of Losken et al. (1991) and Lalikos et al. (1995).

At 18 weeks of age (adulthood), all rabbits were killed by an intracardiac overdose of pentobarbital. Heads were harvested and were cleaned via dermestid beetle action. In order to determine the role of synostosis on increased sutural bone presence, seven calvarial sutures were visually assessed (Fig. 2): coronal (left and right), sagittal, frontal, frontotemporal (left and right), squamosal (left and right), anterior lambdoidal, and posterior lambdoidal sutures. All skulls were viewed under a Wild Heerbrugg microscope equipped with Camera Lucida ($\times$12 magnification). A sutural bone was defined as an island of bone interposed between the two sides of a normally occurring suture (Pucciarelli, 1974; Antón et al., 1992; Schwartz, 1995). Any such structure clearly visible was counted.

It was noted that the data were not normally distributed. Thus, nonparametric statistics were employed. Occurrence of sutural bones among normal in-colony, out-colony, delayed onset, and experimentally immobilized rabbits was compared using a Kruskal-Wallis one-way ANOVA test; occurrence of sutural bones between groups was compared using a Mann-Whitney U-test for two independent samples and a Bonferroni correction for multiple comparisons (Thomas, 1986; Marascuilo and Serlin, 1988). All differences were considered significant if $P < 0.05$. In sutures where it was found that significant differences did exist among groups, a post hoc two-sample binomial test for equal proportions (Marascuilo and Serrin, 1988) was performed between groups to ascertain whether the number of individuals with sutural bones in sutures per group was equal. Differences were considered significant if $P < 0.05$.

RESULTS

Sutural bones were found in every calvarial suture type in every group (Fig. 2; Table 1). Among the four groups, significant differences were found only in the coronal and sagittal sutures, with experimentally immobilized and delayed onset crania having the highest frequencies (Table 1). Mann-Whitney U-tests between groups revealed significant differences between normal out-colony and experimentally immobilized

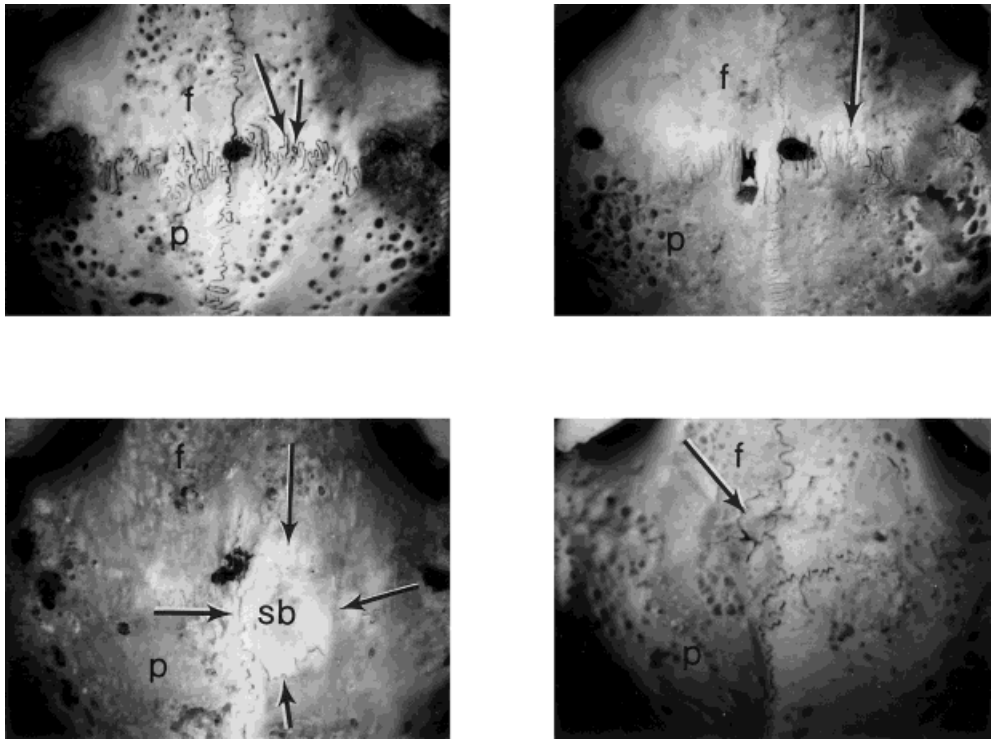

Fig. 2. Dorsal view of rabbit skulls showing representative sutural bones (indicated by arrows). Clockwise from upper left: normal in-colony, normal out-colony, delayed onset, and experimentally immobilized crania. f, frontal bone; p, parietal bone; sb, sutural bone. ×6.

TABLE 1. Average mean number of sutural bone numbers among groups by suture[1]

Suture	Group				Kruskal-Wallis ANOVA	
	NI (N = 14)	NO (N = 12)	DO (N = 25)	EI (N = 20)	χ^2	*P*
Coronal	1.36 (0–5)	1.08 (0–3)	2.37 (0–8)	3.52 (0–13)	9.95*	0.02
Frontal	0.23 (0–3)	0	0.05 (0–3)	0.47 (0–3)	6.86	0.08
Sagittal	1.00 (0–3)	0	0.29 (0–3)	0.79 (0–13)	16.11*	0.01
Anterior lambdoid	0	0	0	0.21 (0–1)	4.81	0.19
Posterior lambdoid	0	0	0.05 (0–1)	0	2.05	0.56
Squamosal	0.25 (0–3)	0.25 (0–3)	0.10 (0–1)	0	1.87	0.60
Frontosquamosal	0	0	0.19 (0–2)	0.10 (0–1)	2.45	0.48

[1] DO, delayed onset; EI, experimentally immobilized; NI, normal in-colony rabbits; NO, normal out-colony. The range of sutural bones present is in parentheses.
* Statistical significance at $P < 0.05$; degrees of freedom = 3.

skulls (EI > NO) (Table 2). There were no significant differences between normal in- and out-colony skulls or between delayed onset and experimentally immobilized skulls (Table 2).

Mann-Whitney U-tests between groups for the sagittal ossicles were not able to show where the significant difference was that appeared among groups in the Kruskal-Wallis one-way ANOVA (Table 2).

As significant differences were found in the coronal and sagittal sutures, a post hoc two-sample binomial test for equal proportions (Marascuilo and Serrin, 1988) was performed between these groups to ascertain whether the number of individuals with sutural bones in the coronal and the sagittal sutures per group was equal. Results are displayed in Tables 3 and 4. The proportion of individual skulls between groups that had

TABLE 2. Comparisons of average mean numbers of sutural bone numbers between groups by suture[1]

	Group comparisons					
Suture	NI/NO	NI/DO	NI/EI	NO/DO	NO/EI	DO/EI
Coronal						
U value[2]	80.0	103.0	86.5	70.5	67.5	223.0
P value[3]	1.00	0.93	0.08	0.58	0.04*	1.00
Sagittal						
U value[2]	42.0	88.5	104.0	114.0	48.0	114.0
P value[3]	0.11	0.37	1.00	1.00	0.23	0.73

[1] DO, delayed onset; EI, experimentally immobilized; NI, normal in-colony rabbits; NO, normal out-colony.
[2] Mann-Whitney U.
[3] All *P* values are adjusted using the Bonferroni correction for multiple comparisons.
* Statistical significance at $P < 0.05$; degrees of freedom = 2.

TABLE 3. Proportions of individuals having at least one sutural bone per group by suture[1]

	Group			
Suture	NI (N = 14)	NO (N = 12)	DO (N = 25)	EI (N = 20)
Coronal	0.64	0.67	0.84	0.80
Sagittal	0.36	0.00	0.08	0.80

[1] DO, delayed onset; EI, experimentally immobilized; NI, normal in-colony rabbits; NO, normal out-colony.

TABLE 4. Comparisons of proportions (p_0) of individuals having at least one sutural bone by suture[1]

	Group comparison					
Suture	NI[1]/NO (p_0)	NI/DO (p_0)	NI/EI (p_0)	NO/DO (p_0)	NO/EI (p_0)	DO/EI (p_0)
Coronal	0.65	0.77	0.73	0.78	0.75	0.82
Z	0.16	1.42	1.03	1.16	0.82	0.35
Sagittal	0.19	0.19	0.62	0.05	0.50	0.40
Z	2.31*	2.19*	2.60*	1.05	4.38*	4.90*

[1] DO, delayed onset; EI, experimentally immobilized; NI, normal in-colony rabbits; NO, normal out-colony.
* Statistical significance at $P < 0.05$.

at least one coronal ossicle was equal. However, proportion comparisons between groups for the sagittal suture revealed that significantly more individuals with experimental immobilization had sagittal ossicles than any other group and that more normal in-colony individuals had sagittal ossicles than did normal out-colony individuals. Thus, the proportion of individual skulls between groups that had at least one sagittal ossicle was uneven, with normal out-colony and delayed onset skulls having a large proportion of individuals with zero sagittal ossicles.

DISCUSSION

Previous studies on the relationship between increased calvarial sutural bone occurrence and altered mechanical loading have found mixed results, some finding a positive relationship (Ossenberg, 1970; Pucciarelli, 1974; Antón et al., 1992; Konigsberg et al., 1993; White, 1996) and others finding none (El-Najjar and Dawson, 1977). The results of this study confirm the hypothesis that increased sutural bone occurrence is influenced more by epigenetic than genetic factors.

The high incidence of coronal sutural bones found in experimentally immobilized crania may be explained by the abnormal coronal sutural growth in these crania. As previous experimental studies have shown, sutures placed under high tension develop bony islands within the membranous portion (Moss, 1957; Young, 1959). In the experimentally immobilized group, the coronal suture is restricted in its normal anteroposterior growth rate, thus presumably placing higher tension than normal upon the suture as the cranial contents expand (Mooney et al., 1994b; Burrows et al., 1995; Lalikos et al.,

1995). This may produce a higher incidence of coronal sutural bones but after the coronal synostosis has begun.

While the delayed onset crania had a higher frequency of coronal sutural bones than the normal groups, the frequency was not statistically significant. The lack of a statistically significant greater number of coronal sutural bones in the delayed onset group may be explained by the growth pattern of the coronal suture. It has been suggested that coronal suture growth in the delayed onset group proceeds through gradual formation of osseous bridges, beginning medially and proceeding laterally, which gradually synostoses the suture (Mooney et al., 1994b). This is in contrast to the immediate cessation of growth at the coronal suture in rabbits with adhesive placed across the suture. The delayed onset group, then, is under tension for shorter periods of time than the experimentally immobilized group, possibly leading to formation of less sutural bones.

Kruskal-Wallis one-way ANOVA tests did show a significant difference among the groups in the average frequencies of sagittal sutural bones, with normal in-colony rabbits having the greatest average frequency and normal out-colony having the lowest. However, Mann-Whitney U-tests with a Bonferroni multiple comparisons correction were not able to extract where this difference was located. This may be due to the strictness of the multiple comparison correction.

CONCLUSIONS

Coronal synostosis in this rabbit colony is a nonsyndromic familial event with the sutural synostosis occurring primary to all other abnormalities of the cranium (Smith et al., 1996) and is inherited as an autosomal dominant condition with variable penetrance (Mooney et al., 1996a). In this rabbit model, familial and experimental coronal synostosis acts as an anteroposterior immobilizer, producing results similar to those found in human crania that are artificially deformed in an anteroposterior fashion (Antón et al., 1992). Sutural bones appear after the growth restriction, not prior to it. Thus, it can be understood that sutural bones are a result, not a cause, of coronal suture synostosis.

While this study confirms the hypothesis that increased sutural bone occurrence is related to epigenetic factors, the influence of genetics on the presence of sutural bones cannot be discounted, as reflected in the equivalency of individuals across groups with coronal sutural bones and the fact that delayed onset crania did not have significantly greater average frequencies of coronal sutural bones than normal crania.

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LITERATURE CITED

Andersson H (1966) Craniosynostosis as a complication after operation for hydrocephalus. Acta Paediatr. Scand. *55:*192–196.

Antón SC, Jaslow CR, and Swartz SM (1992) Sutural complexity in artificially deformed human (*Homo sapiens*) crania. J. Morphol. *214:*321–332.

Bennett KA (1965) The etiology and genetics of wormian bones. Am. J. Phys. Anthropol. *23:*255–260.

Berry AC, and Berry RJ (1967) Epigenetic variation in the human cranium. J. Anat. *101:*361–379.

Berry RJ, and Searle AG (1963) Epigenetic polymorphism of the rodent skeleton. Proc. Zool. Soc. Lond. *140*:577–615.

Brothwell DR (1981) Digging Up Bones. London: Oxford University Press.

Burrows AM (1995) A three-dimensional analysis of craniofacial growth in congenital coronal suture synostosed rabbits. Ph.D. Dissertation, University of Pittsburgh.

Burrows AM, Smith TD, Mooney MP, Losken HW, and Siegel MI (1994) Cranial vault growth in rabbits with nonsyndromic congenital coronal suture synostosis. Am. J. Phys. Anthropol. Suppl. *18:*63.

Burrows AM, Mooney MP, Smith TD, Losken HW, and Siegel MI (1995) Growth of the cranial vault in rabbits with congenital coronal suture synostosis. Cleft Palate Craniofac. J. *32:*235–246.

Cheverud JM, and Buikstra JE (1981) Quantitative genetics of skeletal nonmetric traits in the rhesus macaques on Cayo Santiago. I. Single trait heritabilities. Am. J. Phys. Anthropol. *54:*43–49.

Cohen MM Jr (1977) Genetic perspectives on craniosynostosis and syndromes with craniosynostosis. J. Neurosurg. *47:*886–898.

Cohen MM Jr (1986) The etiology of craniosynostosis. In MM Cohen, Jr. (ed.): Craniosynostosis: Diagnosis,

Evaluation, and Management. New York: Raven Press, pp. 59–80.
Cohen MM Jr (1993) Sutural biology and the correlates of craniosynostosis. Am. J. Med. Genet. *47:*581–616.
Cohen SR, Dauser RC, and Gorski JL (1993) Insidious onset of familial craniosynostosis. Cleft Palate Craniofac. J. *30:*401–405.
Coleman EN, and Foote JB (1954) Craniostenosis with familial rickets. Br. Med. J. *1:*561–562.
Delattre A, Fenart R, and Deblock R (1969) Os wormiens et anomalies suturales dans une collection de crânes de *Pan paniscus.* C. R. Ass. Anat. *144:*1794–1796.
Dembo A, and Imbelloni J (1938) Deformaciones Intencionales del Cuerpo Humano de Caracter Etnico. Buenos Aires: Jose Anesi.
Dorsey GA (1897) Wormian bones in artificially deformed Kwakiutl crania. Am. Anthropol. *10:*169–173.
El-Najjar MY, and Dawson GL (1977) The effect of artificial cranial deformation on the incidence of wormian bones in the lambdoidal suture. Am. J. Phys. Anthropol. *46:*155–160.
Fox RR (1994) Taxonomy and genetics. In PJ Manning, DH Ringler, and CE Newcomer (eds.): The Biology of the Laboratory Rabbit. San Diego: Academic Press, pp. 1–27.
Graham JM Jr, Badura RJ, and Smith DW (1980) Coronal craniostenosis: Fetal head constraint as one possible cause. Pediatrics *65:*995–999.
Green CV, and Fekete E (1932) Some cranial anomalies in mice. Anat. Rec. *53:*187–191.
Greene HS (1933) Oxycephaly and allied conditions in man and in the rabbit. J. Exp. Med. *57:*967–976.
Greene HS, and Brown WH (1932) Hereditary variations in the skull of the rabbit. Science *76:*421–422.
Grüneberg H (1961) Evidence for genetic drift in Indian rats (*Rattus rattus* L.). Evolution *15:*259–262.
Herring SW (1972) Sutures—a tool in functional cranial analysis. Acta Anat. *83:*222–247.
Hess L (1946) Ossicula wormiana. Hum. Biol. *18:*61–80.
Jaslow CR (1989) Sexual dimorphism of cranial suture complexity in wild sheep (*Ovis orientalis*). Zool. J. Linn. Soc. *95:*273–284.
Jaslow CR (1990) Mechanical properties of cranial sutures. J. Biomech. *23:*313–321.
Kellock WL, and Parsons PA (1970) Variation of minor non-metrical cranial variants in Australian aborigines. Am. J. Phys. Anthropol. *32:*409–422.
Kloss JL (1986) Craniosynostosis secondary to ventriculoatrial shunt. Am. J. Dis. Child. *116:*315–317.
Konigsberg LW, Kohn LAP, and Cheverud JM (1993) Cranial deformation and nonmetric trait variation. Am. J. Phys. Anthropol. *90:*35–48.
Koskinen-Moffett L (1986) In vivo experimental model for prenatal craniosynostosis. J. Dent. Res. *65:* (Abstract 980).
Koskinen-Moffett L, and Moffett BC (1989) Sutures and intrauterine deformation. In JA Persing, MT Edgerton, and JA Jane (eds.): Scientific Foundations and Surgical Treatment of Craniosynostosis. Baltimore: Williams & Wilkins, pp. 96–106.
Lalikos JF, Tschakaloff A, Mooney MP, Losken HW, Siegel MI, Losken A, Reitz P, and Wright M (1995) Internal calvarial bone distraction in rabbits with experimental coronal suture immobilization. Effects of overdistraction. Plast. Reconstr. Surg. *96:*689–698.
Losken HW, Mooney MP, Hurwitz DJ, Siegel MI, Losken A, and Zhang LP (1991) Frontal bone advancement and compensatory growth in rabbits with experimental coronal suture immobilization. J. Craniofac. Surg. *2:*86–94.
Marascuilo LA, and Serlin RC (1988) Statistical Methods for the Social and Behavioral Sciences. New York: WH Freeman and Company.
McCarthy JG, and Reid CA (1980) Craniofacial synostosis in association with vitamin D–resistant rickets. Ann. Plast. Surg. *4:*149–153.
Menking M, Wiebel J, Schmidt WT, Ebel KD, and Ritter R (1972) Premature craniosynostosis associated with hyperthyroidism in 4 children with reference to 5 further cases in the literature. Monatsschr. Kinderheilkd. *120:*106–110.
Mooney MP, Losken HW, Tschakaloff A, Siegel MI, Losken A, and Lalikos JF (1993) Congenital bilateral coronal suture synostosis in a rabbit and craniofacial growth comparisons with experimental models. Cleft Palate Craniofac. J. *30:*121–128.
Mooney MP, Losken HW, Siegel MI, Lalikos JF, Losken A, Smith TD, and Burrows AM (1994a) Development of a strain of rabbits with congenital simple nonsyndromic coronal suture synostosis. Part I: Breeding demographics, inheritance pattern, and craniofacial anomalies. Cleft Palate Craniofac. J. *31:*1–7.
Mooney MP, Losken HW, Siegel MI, Lalikos JF, Losken A, Burrows AM, and Smith TD (1994b) Development of a strain of rabbits with congenital simple nonsyndromic coronal suture synostosis. Part II: Somatic and craniofacial growth patterns. Cleft Palate Craniofac. J. *31:*8–16.
Mooney MP, Ashton CE, Siegel MI, Losken HW, Smith TD, Burrows AM, Wenger SL, Siegel B, and Ferrell RE (1996a) Craniosynostosis with autosomal dominant transmission in New Zealand White rabbits. J. Craniofac. Genet. Dev. Biol. *16:*52–63.
Mooney MP, Smith TD, Burrows AM, Langdon HL, Stone CE, Losken HW, Caruso K, and Siegel MI (1996b) Coronal suture pathology and synostotic progression in rabbits with congenital craniosynostosis. Cleft Pal.-Craniofac. J. *33:*369–378.
Moss ML (1954) Growth of the calvaria in the rat. Am. J. Anat. *94:*333–361.
Moss ML (1957) Experimental alteration of sutural area morphology. Anat. Rec. *127:*569–590.
Moss ML, and Young RW (1960) A functional approach to craniology. Am. J. Phys. Anthropol. *18:*281–292.
Ossenberg NS (1970) The influence of artificial cranial deformation on discontinuous morphological traits. Am. J. Phys. Anthropol. *33:*357–372.
Pfeiffer L (1900) Einige Medizinisch Interessante Funde aus dem Merovinger'schen Graberfeld (5. Jahurhundert n. Ch.) in Weimar. Corr.-Blat. Allgem. Arztlichen Vereins Thuringen *29:*426–437.
Pucciarelli HM (1974) The influence of experimental deformation on neurocranial wormian bones in rats. Am. J. Phys. Anthropol. *41:*29–38.
Reddy K, Hoffman H, and Armstrong D (1990) Delayed and progressive multiple suture craniosynostosis. Neurosurgery *26:*442–448.
Richards GD, and Antón SC (1991) Craniofacial configuration and postcranial development of a hydrocephalic child (ca. 2500 B.C.–500 A.D.): With a review of cases and comment on diagnostic criteria. Am. J. Phys. Anthropol. *85:*185–200.
Richtsmeier JT, Cheverud JM, and Buikstra JE (1984) The relationship between cranial metric and nonmetric traits in the rhesus macaques from Cayo Santiago. Am. J. Phys. Anthropol. *64:*213–222.
Schwartz JH (1995) Skeleton Keys. New York: Oxford University Press.
Smith TD, Mooney MP, Burrows AM, Losken HW, and Siegel MI (1996) Postnatal changes in the cranial base in rabbits with congenital coronal suture synostosis. J. Craniofac. Genet. Dev. Biol. *16:*107–117.

Sperber GH (1989) Craniofacial Embryology. London: John Wright and Sons.

Sullivan LR (1922) The frequency and distribution of some anatomical variations in American crania. Anthropol. Papers Am. Mus. Nat. Hist. *23:*203–258.

Thomas DH (1986) Refiguring Anthropology. Prospect Heights, IL: Waveland Press.

Torgersen J (1951) Hereditary factors in the sutural pattern of the skull. Acta Rad. *36:*374–382.

Torgersen J (1954) The occiput, the posterior cranial fossa and the cerebellum. In J Jansen and A Brodal (eds.): Aspects of Cerebellar Anatomy. Oslo: Johan Grundt, Tanum Forlag, pp. 396–418.

Trinkaus E (1978) Bilateral asymmetry of human skeletal non-metric traits. Am. J. Phys. Anthropol. *49:*315–318.

White CD (1996) Sutural effects of fronto-occipital cranial modification. Am. J. Phys. Anthropol. *100:*397–410.

Young RW (1959) The influence of cranial contents on postnatal growth of the skull in the rat. Am. J. Anat. *105:*383–414.